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Cancer in Animals: The 10 Hallmarks

Cancer is **cellular homeostasis lost** — a single body cell escapes the controls on when it grows, specialises and dies. In 2000, Hanahan and Weinberg distilled every cancer into a set of **hallmarks**: capabilities a cell must acquire to become malignant, from driving its own growth to evading death, building a blood supply and spreading. The same hallmarks underlie the lymphomas, mast-cell tumours, osteosarcomas and mammary cancers vets see every week — and they are exactly what modern oncology aims to switch off.

INSIDE THESE NOTES

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Vets & veterinary students

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STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-06-29

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LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Define cancer as a loss of cellular homeostasis, and distinguish benign from malignant neoplasia.
- ✓ Explain the genetic basis: proto-oncogenes/oncogenes vs tumour-suppressor genes, and the multistep model.
- ✓ List and explain the 10 hallmarks of cancer, with an animal example for each.
- ✓ Describe the metastatic cascade and why metastasis determines prognosis.
- ✓ Recognise the common cancers of dogs and cats and how the hallmarks map to therapy.

TL;DR

Cancer is **cellular homeostasis lost** — a single body cell escapes the controls on when it grows, specialises and dies. In 2000, Hanahan and Weinberg distilled every cancer into a set of **hallmarks**: capabilities a cell must acquire to become malignant, from driving its own growth to evading death, building a blood supply and spreading. The same hallmarks underlie the lymphomas, mast-cell tumours, osteosarcomas and mammary cancers vets see every week — and they are exactly what modern oncology aims to switch off.

AT A GLANCE

DEFINITION	Autonomous cell growth from loss of homeostatic control; benign vs malignant neoplasia
GENETIC BASIS	Oncogenes (gain-of-function, 1 hit) + tumour-suppressor loss (2 hits)
KEY GENES	ras, myc (oncogenes); p53, Rb (suppressors)
FRAMEWORK	The 10 hallmarks of cancer (Hanahan & Weinberg)
LETHAL STEP	Metastasis — the invasion-and-spread cascade
DOGS VS HUMANS	Dogs skew to sarcomas; humans to carcinomas
COMMON DOG CANCERS	Lymphoma, mast-cell tumour, osteosarcoma, haemangiosarcoma, mammary
CLINICAL	Cytology/biopsy → grade + stage → hallmark-targeted therapy

01 What cancer is — the one-line version

- **Cancer = cellular homeostasis lost:** a single **somatic** cell escapes the rules on when it grows, specialises and dies, and grows **autonomously**.
- **Neoplasia** = new autonomous growth. **Benign** = stays local, well-differentiated; **malignant (= cancer)** = invades + **metastasises**.
- **A genetic disease of body cells** — driven by mutation, but usually **not inherited** and **not contagious** (rare viral/transmissible exceptions).
- **Not every lump is cancer:** abscess, cyst, granuloma, haematoma and reactive nodes all mimic tumours → **sample, don't guess**.

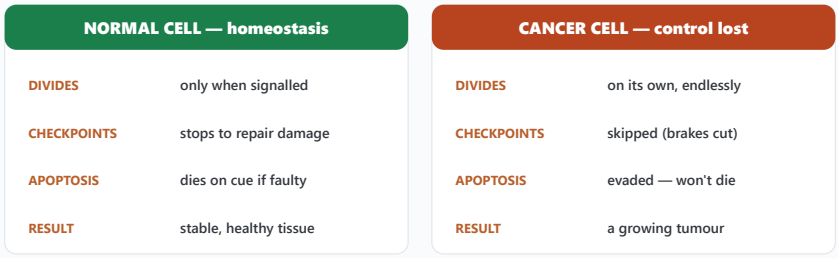

Fig 1 — A normal cell obeys homeostatic controls; a cancer cell has lost every one.

02 The genetic basis — accelerator & brakes

- **Oncogenes** (mutant proto-oncogenes) = a **stuck accelerator**; **one** activating hit is enough (dominant). e.g. *ras*, *myc*.
- **Tumour-suppressor genes** = the **brakes**; cancer needs **both** copies lost (Knudson two-hit). e.g. **p53** (the guardian), **Rb**.
- **Hijacked signal path:** growth factor → RTK → **Ras** → MAP-kinase (Mek/Erk) → **Myc** → cyclins/CDKs → the cell divides.
- **Checkpoints lost:** Rb guards the G1→S gate; **p53** arrests or kills damaged cells. Lose both → damaged cells keep dividing.

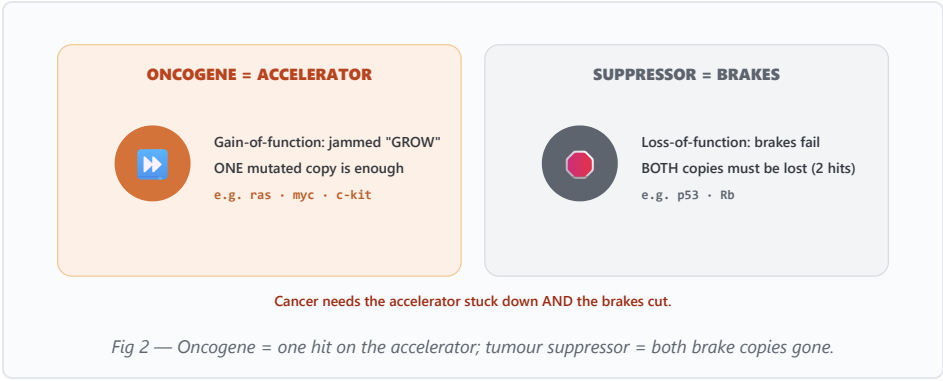

Fig 2 — Oncogene = one hit on the accelerator; tumour suppressor = both brake copies gone.

03 Escaping death (apoptosis)

- **Apoptosis** = programmed cell death; the executioners are **caspases**.
- **Intrinsic (mitochondrial) path** is balanced by the **Bcl-2 family** (pro- vs anti-death); the **extrinsic path** runs via death-receptor signals.
- **Resisting apoptosis is a core hallmark** — Bcl-2 over-expression keeps a doomed cell alive; p53 loss removes the trigger.

04 What causes cancer in animals

- **Multistep + time:** several mutations must stack up → risk rises steeply with age (cancer causes nearly half of deaths in dogs over 10).
- **Breed / inherited risk:** golden retriever, Bernese (haemangiosarcoma, histiocytic sarcoma); boxer, pug (mast-cell); giant breeds (osteosarcoma).
- **Physical / chemical:** UV → squamous cell carcinoma on white cats' ears & nose; chemical carcinogens; chronic inflammation.
- **Oncogenic viruses (very veterinary): FeLV** → feline lymphoma; **papillomavirus** → warts/some SCC; **BLV** → bovine leucosis; Marek's → poultry lymphoma.
- **Some cancers are preventable:** FeLV vaccination, early spaying (mammary), sun avoidance.

05 The 10 hallmarks of cancer

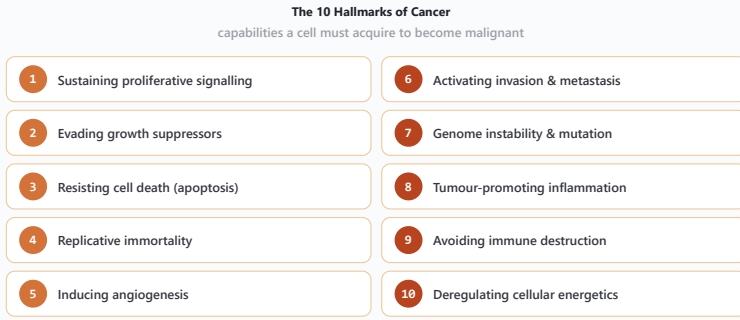

Fig 3 — Six core capabilities + two enabling + two emerging (Hanahan & Weinberg, 2011).

HALLMARK	WHAT THE CELL GAINS
Sustained proliferative signalling	Makes / senses its own growth signals
Evading growth suppressors	Ignores stop signals (Rb, p53)
Resisting cell death	Blocks apoptosis (Bcl-2)
Replicative immortality	Telomerase → unlimited divisions
Inducing angiogenesis	VEGF → a new blood supply
Activating invasion & metastasis	Spreads to distant organs
Enabling: genome instability	Mutations accumulate faster
Enabling: tumour-promoting inflammation	Inflammation feeds growth
Emerging: deregulated energetics	Warburg — glycolysis even with oxygen
Emerging: avoiding immune destruction	Hides from / suppresses immunity

06 Metastasis — the lethal step

- **Why it matters:** metastasis, not the primary lump, is the usual cause of cancer death — local surgery can no longer cure.
- **Cascade:** invade → intravasate (enter a vessel) → circulate/survive → extravasate → **colonise** a distant organ (the hardest step).
- **EMT + enzymes:** cells loosen adhesion (E-cadherin down) and use **MMPs** to dissolve matrix; lung, liver, bone and node are common targets.

Each step is inefficient — most cells die — but a few survivors are enough. In dogs, osteosarcoma → lungs is the classic route.

Fig 4 — Invade → intravasate → circulate → extravasate → colonise.

07 Cancers of dogs & cats

- **Dogs skew to sarcomas** (osteosarcoma, haemangiosarcoma, soft-tissue sarcoma) plus **lymphoma, mast-cell tumour, mammary**.
- **Cats:** lymphoma (FeLV-linked), injection-site sarcoma, oral squamous cell carcinoma.
- **Carcinoma** arises from epithelium; **sarcoma** from mesenchyme (connective tissue).

DOGS	CATS
• Lymphoma	• Lymphoma (FeLV-linked)
• Mast-cell tumour	• Injection-site sarcoma
• Osteosarcoma (→ lungs)	• Oral squamous cell carcinoma
• Haemangiosarcoma (spleen/heart)	• Mammary carcinoma (~90% malignant)
• Mammary tumours	• Skin tumours

Dogs skew to sarcomas (mesenchymal); humans to carcinomas (epithelial).

Fig 5 — The common cancers of dogs and cats.

08 Clinical — diagnose, grade, treat

- **Diagnose by sampling:** FNA cytology first; **biopsy** for architecture, grade & margins. Never diagnose a mass by eye.
- **Grade** (how aggressive the cells look) + **Stage** (how far it has spread, **TNM**) drive both treatment and prognosis.
- **Treat by hallmark:** surgery/radiation (local); chemo e.g. **CHOP** for lymphoma, **carboplatin** for osteosarcoma; **toceranib** (anti-angiogenic TKI); prevention (FeLV vaccine, spay).
- **Prognosis** follows grade + stage, not the word "cancer": lymphoma often remits ~1 yr on CHOP; a low-grade mast-cell tumour is often cured by excision; osteosarcoma with lung mets = months.

RED FLAGS — SAMPLE NOW, DON'T WAIT

Any mass that is rapidly growing, ulcerated, fixed/deep, or at a cat's old injection site — aspirate/biopsy promptly. "Watch and wait" costs stage.

> *Every hallmark a tumour acquires is also a target a therapy can attack — which is why this biology is the backbone of modern veterinary oncology.*
>
> — cancer cell biology, after Cunningham 6e & Hanahan & Weinberg.

KEY TERMS — QUICK GLOSSARY

Neoplasia	New, uncontrolled, autonomous tissue growth; a neoplasm (tumour) may be benign or malignant.
Benign vs malignant	Benign tumours stay local and well-differentiated; malignant tumours invade and metastasise (= cancer).
Proto-oncogene → oncogene	A normal growth-promoting gene; a gain-of-function mutation jams it 'on' (the accelerator). One mutated copy is enough.
Tumour-suppressor gene	A gene that restrains the cell cycle or triggers apoptosis (the brakes); cancer needs both copies lost — e.g. p53, Rb.
Apoptosis	Programmed cell death; resisting it is a core hallmark.
Angiogenesis	Growth of new blood vessels that a tumour induces (via VEGF) to feed itself.
Metastasis	Spread of malignant cells from the primary tumour to distant sites; the main cause of cancer death.
Carcinoma vs sarcoma	Carcinoma arises from epithelium; sarcoma from mesenchyme (connective tissue) — dogs get relatively more sarcomas.

QUICK REVISION — REMEMBER THESE

1. Cancer is the **breakdown of cellular homeostasis**: a single cell escapes the rules that govern when it divides, specialises and dies.
2. It is a **genetic disease of body (somatic) cells**, driven by two opposing gene classes — **oncogenes** (accelerator) and **tumour-suppressor genes** (brakes).
3. Cancer is **multistep** — several mutations must accumulate, which is why risk rises steeply with age.
4. The **10 hallmarks** (Hanahan & Weinberg) are the capabilities a normal cell must acquire to become malignant.

5. **Metastasis** — invasion and spread to distant sites — is what makes cancer lethal, and follows a defined cascade.
6. Animals acquire the same hallmarks: **dogs skew to sarcomas** (osteosarcoma, haemangiosarcoma) plus lymphoma, mast-cell and mammary tumours.
7. Every hallmark is a **drug target** — which is why this biology underpins modern veterinary oncology.

MEMORY AIDS

Gas & brakes — **Oncogenes = a stuck Gas pedal**; **tumour suppressors = cut Brakes**. Cancer needs the gas down *and* the brakes gone.

One hit vs two — Onco**gene** = **one** hit is enough (dominant); suppressor = needs **two** hits (both copies).

-oma trap — Most '**-oma**' tumours are benign (lipoma, adenoma) — but **lymphoma, melanoma, carcinoma, sarcoma** are malignant. Don't trust the suffix.

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **In one sentence, what is cancer at the cellular level?**
2. **Name the two gene classes that drive cancer and their analogy.**
3. **Why does a single mutation rarely cause cancer?**
4. **Give three of the ten hallmarks.**
5. **What makes a tumour malignant rather than benign?**
6. **Why is metastasis so important clinically?**
7. **How do dogs differ from humans in cancer type?**

ANSWERS

1. A somatic cell that has escaped the homeostatic controls on its growth, specialisation and death, and grows autonomously.
2. Oncogenes (a stuck accelerator) and tumour-suppressor genes (cut brakes).
3. Cancer is multistep — several mutations must accumulate, so risk rises with age.
4. e.g. sustaining proliferative signalling, evading growth suppressors, resisting cell death (also angiogenesis, invasion/metastasis, immortality, genome instability, inflammation, immune evasion, deregulated energetics).
5. The ability to invade surrounding tissue and metastasise to distant sites.
6. It is the main cause of cancer death and usually means local surgery alone can no longer cure the disease.
7. Dogs develop proportionally more sarcomas (osteosarcoma, haemangiosarcoma, soft-tissue sarcoma); carcinomas dominate in humans.

WHEN TO REFER OR ESCALATE

- Any rapidly growing, ulcerated, or deeply fixed mass — aspirate/biopsy promptly; don't 'watch and wait'.
- Confirmed malignancy — stage it (imaging ± lymph-node/organ sampling) before surgery, and refer to oncology for chemo/targeted options.
- Pathological fracture or bone pain in a large-breed dog — suspect **osteosarcoma**; image and refer.
- Cat with a firm mass at a previous injection site — suspect **injection-site sarcoma**; plan a wide biopsy + referral.

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